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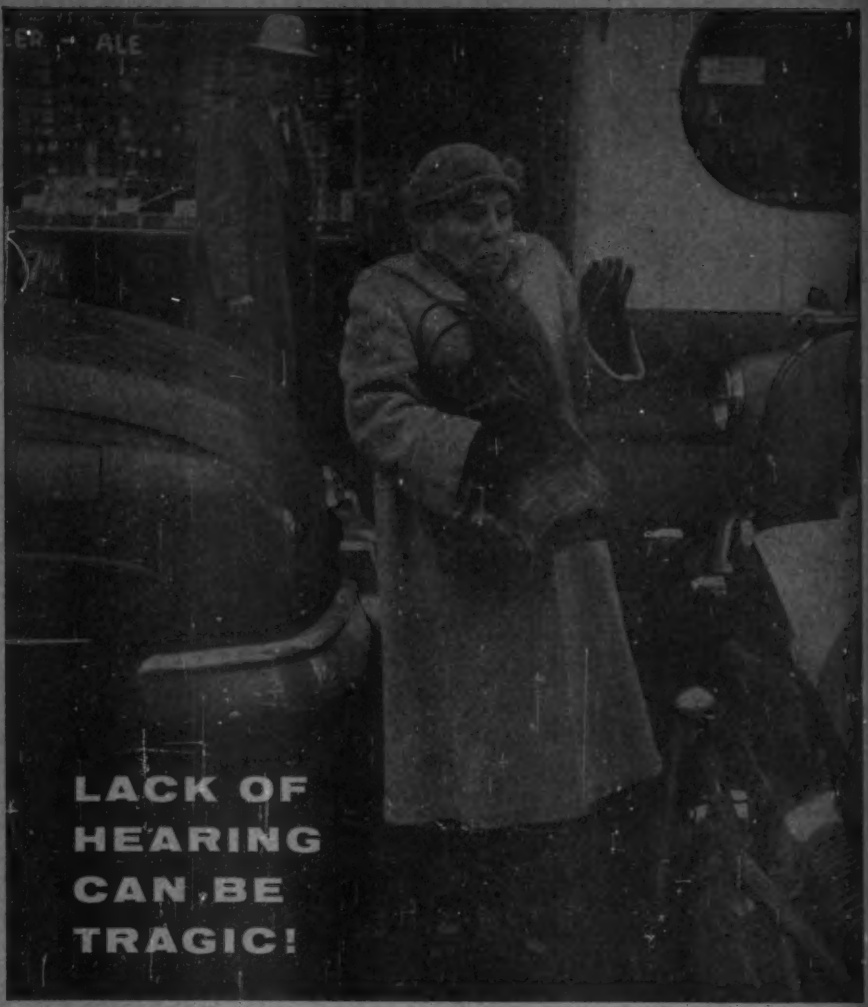

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A HISTOCHEMICAL STUDY OF THE COCHLEA*†

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Recently new methods have been used to obtain further information on the microscopic structure of the labyrinth. The dissecting microscope and the phase contrast microscope have been used by De Vries[1] (1949) and Hilding[2] (1952) for the study of the origin and insertion of the tectorial membrane, and by Katsuki and Covell[3] (1953) and Engström and Wersäll[4] (1953) for an analysis of the finer cell structure of the organ of Corti. The electron microscope was employed by Engström and Wersäll[5] (1953) in their study of the sensory cells, supporting cells and nerve endings. The preparation of the tissue by freezing and drying has been employed by Vilstrup[6] (1950), who examined the cupula and the subcupular space of the cod. By means of ultraviolet microscopy, Hamberger and Hydén[7] (1945) carried out investigations on nuclear and cytochemical changes of the cochlear nerve cells. Histochemical methods showed selective staining of the otolithic membranes, the cupulae and the tectorial membrane (Wislocki and Ladman[8] 1954). Radioactive sulfur (S^{35}), injected in rats, was detected by autoradiography in the cupula, the tectorial membrane and the otolithic membrane of the utricle[9] (Bélanger 1953).

This paper reports histochemical studies of the cochlear duct of the bat after freezing and drying (Gersh[10,11] 1932, 1948, Gersh and Stephenson,[12] 1954). The bat was found very suitable for

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such studies for several reasons: *1*, The internal ear is readily removed with great rapidity and is of such small size as to be very well fixed by freezing and drying. Were the structure larger, the ice crystals which form during the freezing would be too large, and disruptive to the delicate structures, for detailed cytological analysis. *2*, The bony plates of the os petrosum are so thin in the bat that it is possible to cut thin paraffin sections of the internal ear without decalcification. It is further possible to cut polyethelene glycol-embedded sections with the freezing microtome for demonstration of lipids. *3*, The rapidity of fixation reduces to a minimum the post-mortem changes which take place during fixation by immersion in a chemical fixative, or even by perfusion.

In addition many effects of the fixatives themselves are avoided by the freezing-drying method employed in this study. The methods of fixation usually employed will result in a more or less marked displacement of easily soluble and highly diffusible substances. Polysaccharides, proteins, lipids and enzymes are very well preserved by the freezing-drying technique of fixation. As the polysaccharide-containing proteins of the tissues are preserved with only minimal denaturation by this method, it is possible to make qualitative studies of the solubility of some of these components by the use of various extractives (Gersh and Catchpole,[13] 1949).

Various histochemical staining reactions were employed in this study. These included the procedure for polysaccharides, developed by McManus[14] (1946), Lillie[15] (1947) and Hotchkiss[16] (1948), metachromatic staining*, reactions for neutral fat, plasmalogen (a fat with aldehyde groups) and alkaline phosphatase.

MATERIALS AND METHODS

Bats (Myotis lucifugus and subspecies) caught in caves during hibernation were used. The animals were killed by decapitation. The mandible was removed and the bulla exposed. After opening the bulla, the cochlea, with the vestibular apparatus, was easily lifted out. This procedure usually took one to two minutes after the death of the animal.

The specimen was immediately frozen in isopentane at approximately —155°C., and later dehydrated in vacuo at about —30°C.

* Structures stained with toluidine blue may appear blue or red to purple. The former are said to be stained orthochromatically; the latter are said to be metachromatic.

over a period of three to seven days. The tissue was infiltrated with paraffin (m.p. 62°C.) in vacuo for 15 minutes and embedded. For the phosphatase reaction, paraffin with a melting point at 51°C. was used. Other specimens were transferred from the vacuum tube to absolute alcohol for one day, then to 95 percent alcohol for another day. After decalcification in a solution of 0.5 per cent

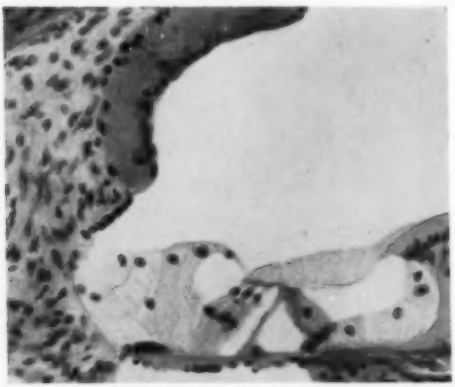

Fig. 1. Organ of Corti of the bat. Section prepared by the freezing-drying method, stained with hematoxylin-eosin—X 450.

hydrochloric acid in 80 per cent alcohol for about a week they were embedded in celloidin.

Decalcified sections embedded in celloidin were stained with hematoxylin-eosin and by the alcoholic periodic acid-leukofuchsin method described by Hotchkiss[16] (1948). Undecalcified, paraffin-embedded sections were deparaffinized with xylol or petroleum ether, coagulated with absolute alcohol for about 16 hours and stained for glycoproteins after the Hotchkiss procedure. Such sections were used for the study of the total periodic acid-leukofuchsin-reactive material. They were compared with other sections which have been treated before staining with saliva, N/10 hydro-

chlorie acid and various buffers with their respective pH: Acetate pH 2.9, 3.2, 4.2; phosphate pH 5.5, 6.0, 7.0; borate pH 8.9.

To determine the glycolipid fraction of the total reactive substances sections were deparaffinized in petroleum ether, immersed in equal parts of chloroform and methanol at 60°C. for 16 hours, denatured in ethanol for about 16 hours and treated by the

Fig. 2. Organ of Corti of the bat. Frozen-dried, stained by the periodic acid-leukofuchsin method of Hotchkiss—X 1230.

The condensed fibers of the basilar membrane are deeply stained. The inner hair cells, the Deiters' cells and the Böttcher's cells contain Hotchkiss positive material, which is extractable by dilute hydrochloric acid, various buffers and saliva. The surface of the Hensen's cells is covered by Hotchkiss positive filiform processes.

Hotchkiss method. For metachromatic staining, the paraffin was removed by petroleum ether, and the solvent was allowed to evaporate. The sections were covered with a few drops of 0.05 per cent aqueous toluidine blue (pH 5.6) and observed directly.

For demonstration of neutral fat and plasmalogen the tissue was prepared according to the method of Hack[17] (1952). The frozen-dried specimens were infiltrated in the vacuum tube with polyethelene glycol "200" for 30 minutes without breaking the vacuum.

The tissue was then transferred to "400" polyethelene glycol, and kept at a temperature of 4°C. Sections were cut with a freezing microtome, Sudan-black B was used for the staining of neutral fat. The plasmal reaction was carried out as described by Feulgen and Voit[18] (1924).

The calcium phosphate method of Gomori[19] (1939) was applied for demonstration of the sites of alkaline phosphatase activity in the tissue. The slides were incubated for one, three and 12 hours.

Fig. 3. Basilar membrane of the organ of Corti (lower coil). Frozen-dried, stained by the Hotchkiss method—X 1400.

The fibers of the basilar membrane are Hotchkiss positive and embedded in small amounts of interfibrillar substance, which is less deeply stained. Both components are not extractable by dilute hydrochloric acid, various buffers and saliva.

RESULTS

Membrana basilaris. The basilar membrane of the lower coil of the cochlear duct consists of two well defined zones, the inner zone (zona arcuata) and the outer zone (zona pectinata) as seen in Figs. 1 and 3. Radially arranged thick fibers run through both zones. All fibers of the zona pectinata are radially arranged while fibers in the predominant thickening of the zona arcuata run in the direction of the cochlear duct. Toward the apex of the cochlea the thickenings of both zones disappear gradually. In the upper coil the basilar membrane is thin and consists mainly of radially arranged fibers (See Fig. 4).

All fibers of the basilar membrane were intensely stained by the Hotchkiss method (See Figs. 2 and 3). Small amounts of interfibrillar substance were less deeply stained. After treatment with the various extractives no change of stainability of both components

of the membrane was noted. With toluidine blue the fibers and the ground substance were stained metachromatically reddish-purple.

The scanty protoplasm of the mesothelial cells on the scala tympani surface of the basilar membrane showed a pink color by the Hotchkiss method.

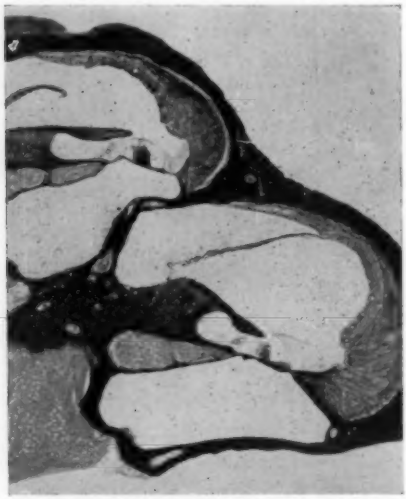

Fig. 4. The lower and the upper coil of the cochlear duct of the bat. Frozen-dried, stained by the Hotchkiss method—X 150.

The Deiters' cells are more intensely stained in the upper coil. Hotchkiss stainable granules in the stria vascularies are more numerous in the upper coil.

Ligamentum Spirale, Limbus Spiralis. The fibers of the ligamentum spirale and the limbus spiralis were stained deep red after the Hotchkiss procedure (See Figs. 4, 5 and 11). The connective tissue cells and the ground substance were stained pink. Hydrochloric acid (N/10), the various buffers and saliva did not change the stainability of the connective tissue components. When the sections were incubated in a hot chloroform-methanol solution

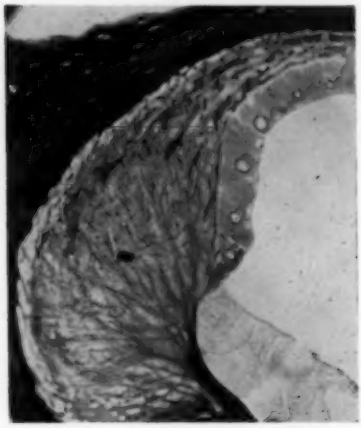

Fig. 5. The spiral ligament and the stria vascularies of the cochlear duct of the bat. Frozen-dried, stained by the Hotchkiss method—X 425.

The connective tissue fibers are intensely stained, the ground substance, the connective tissue cells and the cytoplasm of the stria vascularis reveal a pink color. This Hotchkiss positive material was not affected by dilute hydrochloric acid, various buffers and saliva.

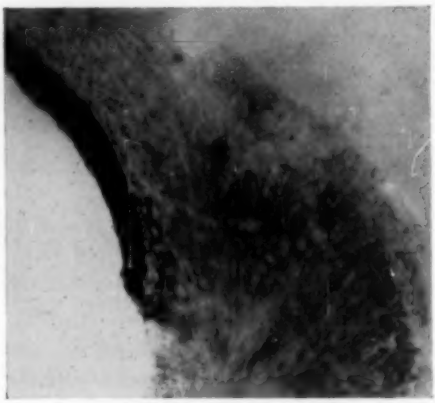

Fig. 6. The spiral ligament and the stria vascularis of the cochlear duct of the bat. Frozen-dried, undecalcified, embedded in polyethelene glycol, stained for plasmalogen—X 125.

Plasmalogen is demonstrated in the ground substance and the cells of the spiral ligament, and in the cytoplasm of the stria vascularis. Connective tissue fibers and nuclei are plasmal negative.

a very slight decrease of stainability of the ground substance was noted. The fibrous structure of the ligamentum spirale and the limbus spiralis appeared reddish-purple, the cells and the ground substance more purple after metachromatic staining. While the thick fibers were plasmal negative, the ground substance and the connective tissue cells of the ligamentum spirale were plasmal positive (See Fig. 6). Comparable components of the limbus spiralis were less intensely stained for plasmal.

The "Huschke's teeth", consisting mainly of condensed fibers, stained deep red by the Hotchkiss method (See Figs. 7 and 8). The cytoplasm of the epithelial cells of the limbus spiralis, which continue in the inner sulcus cells, did not stain by the Hotchkiss or by the plasmal reaction.

The Sensory Cells of the Organ of Corti. The outer hair cells with their cuticles were not stained by the Hotchkiss method, toluidine blue for metachromasia, the plasmal or phosphatase reactions. The reticular membrane was also negative; however, the cytoplasm of the inner hair cells revealed a pale pink color by the Hotchkiss method (See Figs. 2 and 10). After immersion in chloroform-methanol a decrease in stainability was not noted, but the pink Hotchkiss stain could not be observed when the sections were pretreated with N/10 hydrochloric acid, the various buffers and saliva before or after alcoholic denaturation.

The cytoplasm of the inner hair cells stained very faintly by the plasmal reaction. Metachromasia and alkaline phosphatase activity were not apparent in the inner hair cells.

Supporting Cells, Böttcher's Cells, Inner and Outer Sulcus Cells. The cytoplasm of the Deiters' and Bötteher's cells stained pink by the Hotchkiss method (See Figs. 2 and 10). The stain was more intense in the cells of the upper coil of the cochlear duct (See Fig. 4). After treatment with the various extractives the cytoplasm of

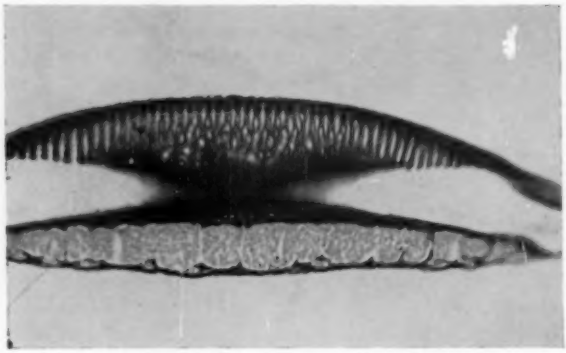

Fig. 7. Huschke's teeth of the limbus spiralis (bat). Frozen-dried, Hotchkiss method—X 300.

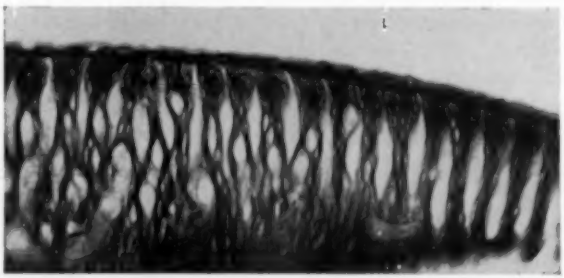

Fig. 8. Huschke's teeth of the limbus spiralis (bat). Frozen-dried, Hotchkiss method—X 925. Huschke's teeth consist mainly of condensed deeply stained fibers.

these cells could not be stained. Metachromasia was not observed in these cells. When Hack's method for plasmalogen was used, the cytoplasm of the Deiters' and Böttcher's cells stained faintly. The other supporting cells, as well as the inner and outer sulcus cells, did not exhibit a positive reaction with the histochemical reactions employed in this study. The outer sulcus cells in the upper coil

Fig. 9. Membrana tectoria (bat). Frozen-dried, Hotchkiss method—X 1625.

The fibers are intensely stained, the interfibrillar substance reveals a pink color. The membrane is attached to the vestibular lip of the limbus spiralis by a deeply stained homogenous substance.

Fig. 10. Membrana tectoria (bat). Frozen-dried Hotchkiss method—X 1135.

Note marginal network ("Randfasernetz") on the upper surface of the membrane and Hotchkiss positive filiform processes on the surface of the Hensen's cells.

protruded into the connective tissue of the spiral ligament in a few sections (See Fig. 15).

Membrana Tectoria. The numerous fibers of the tectorial membrane were deeply stained by the Hotchkiss method and were separated by pink-staining material (See Fig. 9). The various extractives did not alter this pattern of staining. The fibers gave an intense metachromatic reaction with toluidine blue (reddish-purple), while the material between them appeared purple. In contrast to the spiral ligament and the limbus spiralis the interfibrillar material of the tectorial membrane was negative for plasmalogen, and was only very faintly stained with eosin.

The general direction of the fibers of the membrane is radial to the axis of the cochlea with a marked deviation towards its lower surface. Approaching the lower surface the fibers bend in the radial direction. Branches follow the same general direction. The fibers are condensed at the limbal portion of the membrane. The limbal portion is attached to the underlying epithelial cells of the limbus by a homogenous material stained red by the Hotchkiss method and reddish-purple with toluidine blue for metachromasia.

The tectorial membrane was attached with its lower surface to the organ of Corti. A marginal network ("Randfasernetz" of Deiters) was seen in nearly all sections, and had the same staining qualities as the fibers of the membrane (See Fig. 10). It covered mostly one-third to one-half of the upper surface of the membrane ("upper surface": extending from the outer margin to the vestibular lip of the limbus spiralis). Hotchkiss positive filiform processes with the same staining features as the marginal network were seen on the surface of the Hensen's cells (See Figs. 2 and 10). In some instances a pink-reddish stripe covered the surface of the outer sulcus cells and the surface of a layer of two or three inner sulcus cells adjacent to the organ of Corti.

Stria Vascularis. The cytoplasm of the stria cells stained pink when the Hotchkiss method was applied (See Fig. 11). Hotchkiss positive, very thin threads in the axial direction of the cells were present in the cytoplasm. Small granules stained intensely after the Hotchkiss procedure and exhibited metachromasia. They were

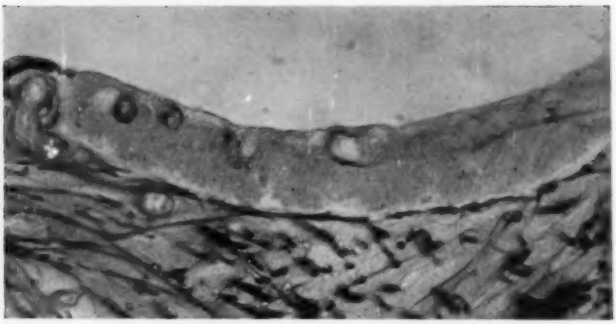

Fig. 11. Stria vascularis of the lower coil of the cochlear duct (bat). Frozen-dried, Hotchkiss method—X 1035.

Only a few Hotchkiss stainable granules are seen in the cytoplasma of the stria cells. The fibers in the connective tissue adjacent to the stria vascularis stain deep red.

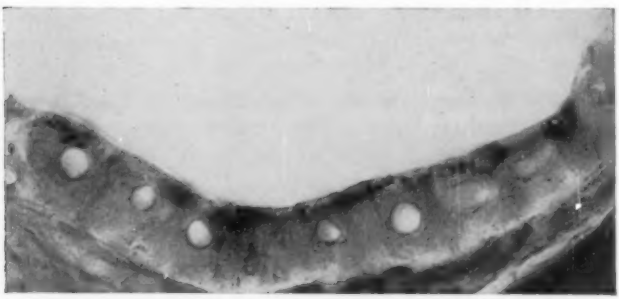

Fig. 12. Stria vascularis of the upper coil of the cochlear duct (bat). Frozen-dried, Hotchkiss method—X 940.

Note aggregations of the Hotchkiss stainable granules close to the upper surface of the cells, and the sharply defined basement membranes around the capillaries.

visible mostly in the upper half of the cells around the nuclei, which are localized closely under the free surface of the epithelium. The granules were most numerous in the upper coil of the cochlear duct (See Fig. 12).

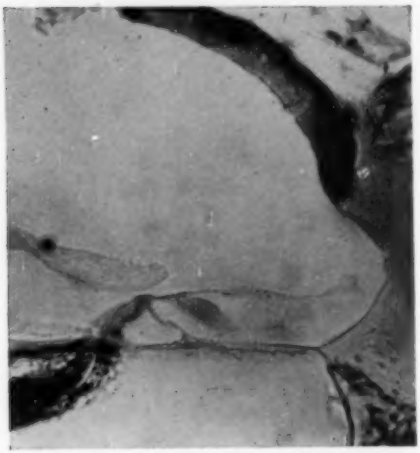

Fig. 13. Cochlear duct of the bat. Frozen-dried, undecalcified, alkaline phosphatase reaction—X 400.

Positive phosphatase reaction in the basal part of the stria vascularis adjacent to the connective tissue. In the control without glycerol phosphate, the cells of the stria vascularis were unstained.

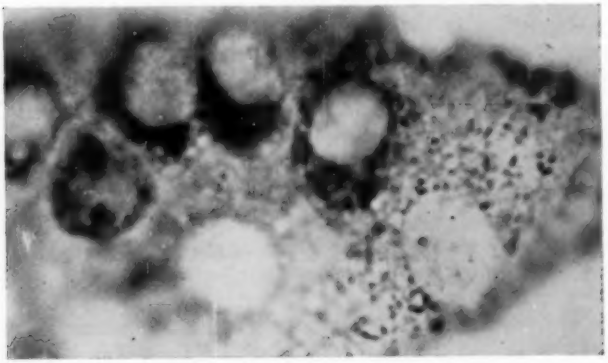

Fig. 14. Reissner's membrane (bat). Tangential section. Frozen-dried Hotchkiss method—X 3450. Numerous minute granules are stained intensely.

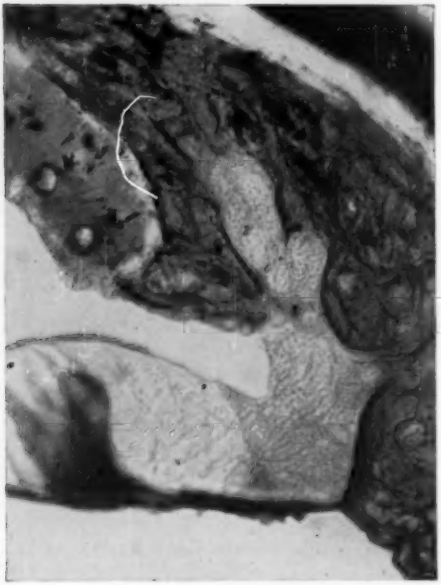

Fig. 15. Outer sulcus cells in the upper coil of the cochlear duct (bat). Frozen-dried, Hotchkiss method—X 1075.

Note pale staining cells protruding in the connective tissue of the spiral ligament.

The Hotchkiss positive material in the stria vascularis was not extractable with dilute hydrochloric acid, the various buffers and saliva. The cytoplasm of the cells revealed a rather strong reaction for plasmalogen (See Fig. 6). When the alkaline phosphatase reaction was applied, incubation of the slides for one hour, as well as for three and twelve hours, showed a positive reaction in the basal part of the stria vascularis without major changes in its localization during the various incubation periods (See Fig. 13).

The Hotchkiss procedure revealed very sharply defined basement membranes around the capillaries of the stria vascularis (See Fig. 12). The membranes were not affected by the various extractives. Yellow-brownish pigment granules were found irregularly dis-

tributed in the stria vascularis. A layer of small epithelial cells between the stria and the Hotchkiss negative outer sulcus cells stained very similar to the stria epithelium.

Reissner's Membrane. The cytoplasm of the epithelial cells of Reissner's membrane stained pink by the Hotchkiss method (See Figs. 4 and 14). The scanty cytoplasm of the mesothelial cells was similarly stained. Numerous minute granules stained intensely red by the periodic acid-leukofuchsin reaction and exhibiting metachromasia were found in the cytoplasm of the epithelial cells (See Fig. 14). These granules were more frequently seen in the regions of insertion of the membrane. After treatment with N/10 hydrochloric acid, the various buffers and saliva, the Hotchkiss positive material in the membrane, did not change in stainability. When the plasmal reaction was carried out, the cytoplasm of the epithelial cells stained faintly. Small vacuoles of various sizes were occasionally observed in the cytoplasm of the epithelial cells. Lipids were not demonstrated in these cells. Pigment granules similar to those in the stria vascularis were found in varying amounts in the epithelium.

A homogenous basement membrane separating the epithelial cells from the mesothelial cells was intensely stained after the Hotchkiss procedure. The stainability was not altered by dilute hydrochloric acid, the various buffers and saliva.

DISCUSSION

According to Gersh and Catchpole[13] (1949) variations in the consistency of ground substance are responsible for its plasticity. The freezing-drying method of fixation permits qualitative studies of the solubility of some glycoprotein components at the microscopic level. When tested with dilute hydrochloric acid and buffers of differing pH, the glycoprotein of the connective tissue of the spiral ligament and the limbus spiralis, and of the tectorial and Reissner's membrane was not affected. These features indicate that the ground substance is firm and highly aggregated.

Certain classes of lipids like kerasin, lecithin phosphatide and others may give a positive periodic acid-leukofuchsin reaction. (Hack, cited by Gersh and Catchpole[13] 1949). There was a slight decrease of stainability of the ground substance in the spiral liga-

ment and the limbus spiralis after incubation of the sections in a hot chloroform-methanol solution which indicates that some lipids of the varieties mentioned may be present in these sites. The ground substance of these structures was stained by Hack's method for visualizing plasmalogen, indicating the presence of acetal phosphatides of unknown type.

The tectorial membrane is described as containing a gelatinous substance of jelly-like nature, rich in water (Kolmer[20] 1927). The specific gravity is relatively low (Hardesty[21] 1915). The Hotchkiss procedure revealed a strongly positive reaction of its fibers, and the interfibrillar substance was stained pink. As the stainability was not affected by the extractives, it is suggested that the glycoprotein of the tectorial membrane is highly aggregated. Plasmalogen was not found in the interfibrillar substance of the tectorial membrane. In contrast to the spiral ligament and the limbus spiralis eosin stained the ground substance only very slightly. These findings indicate a marked difference of the chemical composition between the Hotchkiss positive material of the tectorial membrane and that of the spiral ligament and the limbus spiralis.

The experiments of Wislocki and Ladman[8] (1954) did not suggest a sulfated mucopolysaccharide as a component of the tectorial membrane. A moderate reaction for protein-bound sulfhydryl and disulfide groups was reported by these authors. Bélanger[9] (1953), who injected radioactive sulfur in immature rats, found a high concentration of S^{35} in the tectorial membrane indicating the synthesis of an organic sulfur compound. When stained with toluidine blue, metachromasia of the fibers and the interfibrillar substance of the tectorial membrane was observed in our experiments. Until recently metachromasia was believed to be specific for the presence of sulfuric esters of large molecular size. Michaelis[22] (1947) proposed that highly polymerized carbohydrates produce metachromatic effects by virtue of their carboxyl groups. Bélanger[3] (1953), using immature rats as experimental animals, reported the persistence of metachromasia in the tectorial membrane after incubation of the slides with hyaluronidase.

In our study the tectorial membrane was attached with its lower surface to the organ of Corti. Its insertion has been the subject of controversial opinions. Using phase contrast microscopy De

Vries[1] (1949), Hilding[2] (1952), Katuski and Covell[3] (1953) offered some evidence that the marginal network ("Randfasernetz" of Deiters) usually covers the Hensen's cells, but is torn loose during the preparation of the tissue and thrown back on the upper surface of the tectorial membrane. Our sections showed that the marginal network and the small filiform processes on the surface of the Hensen's cells had the same staining properties when subjected to the Hotchkiss procedure. This finding may be regarded as support for the opinion that the tectorial membrane is attached to the Hensen's cells in vivo.

According to Held[23] (1926), Kolmer[20] (1926) and others the limbal portion of the tectorial membrane is attached to the underlying epithelial cells by a cement substance. This substance has the same staining properties as the marginal network of the membrane and the filiform processes on the Hensen's cells.

The stria vascularis is generally believed to secrete the endolymph, or at least to produce certain substances which pass into the endolymph. The close relationship between the epithelial cells and the capillaries suggest a secretory activity. Morphological findings (Von Fienandt and Saxén[24] 1936), pharmacological experiments with atoxyl (Miyamoto[25,26] 1931, 1932, Ozeni[27] 1937, Rüedi[28] 1951) and pathological observations in cases of angiosclerotic inner ear degeneration (Saxén[29] 1951) are regarded as support for this assumption.

Our study revealed certain histochemical features of the stria vascularis which were quite different from those of other epithelial cells of the cochlear duct. In contrast to the Hotchkiss positive material in the Deiters' and Böttcher's cells the substances in the stria cells were not extractable with dilute hydrochloric acid, various buffers and saliva. Hotchkiss stainable granules, revealing metachromasia, were more frequently seen in the vicinity of the nuclei localized closely under the free surface and at the surface itself.

The most striking difference between the stria cells and the other epithelial cells was apparent when the reaction for alkaline phosphatase was applied. Sites of phosphatase activity were found in the basal part of the stria vascularis. It was not possible to decide if the reactive material was localized only in the small, star-like "deeper" cells of the pseudo-stratified stria epithelium (Iwata[30]

1924). The sites of activity were obviously unrelated to the capillaries of the stria, as they were demonstrated in the opposite pole of the epithlium adjacent to the connective tissue of the spiral ligament. Connective tissue is known to be involved in the storage and transport of metabolites. It may be suggested that phosphate esters passing from the connective tissue into the stria vascularis are hydrolized by the alkaline phosphatase found in the basal part of the stria cells.

According to Von Fienandt and Saxén[31] (1936) and Saxén[29] (1951) the cells of the sulcus spiralis externus are partly responsible for the reabsorption of the endolymph and for the removal of breakdown products of the cochlear duct. The outer sulcus cells are described as being provided with long protoplasmic processes extending into the connective tissue of the spiral ligament and may function as phagocytic groups (Von Fienandt and Saxén[31] 1936). Only in a few sections were cells similar to those as described by Saxén[29] (1951) seen in the cochlear duct of the bat. The histochemical reactions for glycoprotein, plasmalogen and alkaline phosphatase were consistently negative in the outer sulcus cells.

Kolmer[32] (1911) and Iwata[33] (1925) assumed a functional difference between the outer and inner hair cells because of their distinct morphological differences. The outer hair cells did not stain with histochemical reactions for glycoprotein, plasmalogen, neutral fat and alkaline phosphatase; however, the inner hair cells stained pink by the Hotchkiss method. The Hotchkiss stained material was readily extracted with dilute hydrochloric acid, the various buffers and saliva. In contrast to the negative finding in the outer hair cells, the cytoplasm of the inner hair cells contained plasmalogen.

It has been proposed by Engström and Wersäll[34] (1953) that the Deiters' and Hensen's cells may be regarded as a nutritive aid system for the sensory cells of the organ of Corti. In contrast to the other supporting cells, only the Deiters' cells stained after the Hotchkiss procedure. The solubility tests indicate the presence of a readily soluble glycoprotein or polysaccharide similar to that found in the inner hair cells. These substances stained more intensely in the upper coil of the cochlear duct than in the lower coil. Similarly, it is interesting that glycogen found in the stria cells of the human fetus is still visible in the stria epithelium of the upper coil when

this substance has already disappeared from the epithelium of the lower coil of the cochlear duct (Kolmer[20] 1926).

Using electron microscopy Engström and Wersäll[24] (1953) described a "rich system of minute filiform processes" on the outer surface of the Hensen's cells and the phalanx of the Deiters' cells. They compared this structure with the striated border of the intestine which is regarded as a highly differentiated resorptive organ. The striated border of the intestine, as well as of the proximal tubules of the kidney, exhibits a strong alkaline phosphatase reaction; but no phosphatase activity was observed in the surface region of the Hensen's cells in our experiments.

Kolmer[20] (1926) reported globules of neutral fat in the Hensen's cells of some mammals (guinea pig and squirrel) and occasionally in humans. Katsuki and Covell[3] (1953) using phase contrast microscopy found highly refractile bodies of varying size in the Hensen's cells of the guinea pig, but the authors doubt that all these globules contain fat. In our study the histochemical reaction for neutral fat, and plasmalogen was negative in the Hensen's cells of hibernating bats.

The Böttcher's cells revealed the same histochemical reactions as the Deiters' cells. The outer sulcus cells which cover the Böttcher's cells did not exhibit histochemical reactions by methods employed in this study. The physiological rôle of the Böttcher's cells is as yet unknown.

In all those epithelial cells revealing a positive Hotchkiss reaction (stria cells, Deiters' cells, Böttcher's cells, inner hair cells) plasmalogen was found, also. According to Haek[17] (1925) plasmalogens may be considered as metabolites linked together by low energy bonds. These bonds can be broken to release substances which may thereby become available in metabolic processes.

According to Wolff (quoted by Engström and Wersäll,[5] 1953) the fibers of the outer pillar cells originate from fibers of the basilar membrane. Engström and Wersäll[5] (1953) did not find any connection between the fibers of the pillar cells and those of the basilar membrane using the electron microscope. The Hotchkiss method and metachromatic staining revealed a positive reaction of the fibers of the basilar membrane. The fibers of the pillar cells, however, were negative.

In Reissner's membrane, the membrana propria was described by Gottstein[35] (1872) as separating the epithelial cells from the mesothelial cells. In the bat, this membrane is very thin. It stained intensely by the Hotchkiss method and was not extractable with dilute hydrochloric acid, various buffers and saliva.

Granules apparently containing glycoproteins were seen in varying amounts in the epithelial cells of Reissner's membrane.

SUMMARY.

1. The cochlea of bats caught during hibernation was found to be suitable for detailed morphological and histochemical study after fixation by freezing and drying. The histochemical methods included the Hotchkiss procedure (with solubility tests), metachromasia, and staining procedures for alkaline phosphatase, neutral fat and plasmalogen.

2. Connective tissue fibers were deeply stained by the Hotchkiss procedure. The ground substance of the basilar membrane, the ligamentum spirale and the limbus spiralis contained very insoluble glycoproteins. Plasmalogen was found in the ground substance of the connective tissue of the spiral ligament and the limbus spiralis.

3. The outer hair cells of the organ of Corti were not stained by the histochemical methods employed. A water-soluble Hotchkiss-positive substance and plasmalogen were found in the inner hair cells.

4. Compounds apparently similar to those found in the inner hair cells were present in the Deiters' and Böttcher's cells. The other supporting cells as well as the inner and outer sulcus cells were, on the other hand, negative by the histochemical methods employed.

5. The fibers of the tectorial membrane stained deeply with the Hotchkiss method, but no plasmalogen was found there. A marginal network reacting histochemically like the fibers of the membrane was seen in nearly all sections. Hotchkiss positive filiform processes, with the same staining features as the marginal network and the fibers, were observed on the surface of the Hensen's cells.

6. Hotchkiss positive granules exhibiting metachromasia were demonstrated in the pink stained cytoplasm of the stria epithelium. The Hotchkiss positive material in the stria cells was not extractable

with N/10 hydrochloric acid, buffers of various pH and saliva. The cytoplasm of the cells revealed a positive reaction for plasmalogen. Alkaline phosphatase activity was found in the basal part of the stria vascularis.

7. Hotchkiss positive granules exhibiting metachromasia were found in the cytoplasm of the epithelial cells of Reissner's membrane. The membrana propria stained intensely by the periodic acid-leukofuchsin reaction and was not extractable by the reagents employed.

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DIFFUSE EXTERNAL OTITIS: PATHOGENESIS AND TREATMENT No. 2.*†

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In a previous report[1] based on clinical and pathological data available at that time, a new concept of the pathogenesis of diffuse external otitis was presented. On the basis of this concept, certain general and specific principles of therapy were enumerated. In the ensuing five years, further pathological and clinical data have accumulated. It is my purpose to modify the original concept in view of these developments.

An important contribution found in a review of the literature is that of Collins.[2] This study described an evaluation of the effect of water, high temperatures and humidity on the skin of the external auditory canal. Pertinent observations were made, using human volunteers, on the distribution of eccrine sweat glands around the external ear. The author concluded that an accumulation of sweat in the ear canal was probably not a factor in the production of otitis externa in hot climates. The works of O'Brien,[3] Shelley,[4] Lobitz[5] and Sulzberger and his associates[6,7] are concerned with the physiology and pathology of sweat glands and have been helpful in formulating our opinions.

It is important to appreciate the manner in which the healthy skin of the ear canal functions, in order to understand the pathological changes which occur in diffuse external otitis; therefore, let us examine the anatomy and histology of the external ear. For purposes of this presentation, we may consider the external ear as divided into two parts: *1.* The auricle and the periauricular skin, and *2.* The external auditory canal and the tympanic membrane.

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The auricle and external auditory canal show great individual variations in size and shape. In some cases, the tragus and antitragus form a marked obstruction at the entrance of the ear canal, while the long axes of the two canals form an obtuse angle. Some lumina are wide and straight without any constriction and thus allow easy access to fluids, while others are narrow and present curvatures which serve to make the entrance or removal of fluids difficult.

The external auditory canal is composed of *a.* a cartilaginous canal and *b.* a bony canal. The cartilaginous canal is pliable and flexible as a result of the structure of the cartilage and the vertical fissures of Santorini in the anterior wall. The narrowest part of the ear canal, the isthmus, is formed by the marked convexity of the floor and anterior wall of the osseous canal and lies just medial to the junction of the osseous and cartilaginous portions. The canal widens abruptly at the drum to form the sulcus of the tympanic membrane.

The skin of the auricle is provided with a distinct subcutaneous layer on its posterior convex surface only. It carries a few small hairs with sebaceous glands of considerable size. These are particularly evident in the concha. The sweat glands are of the eccrine type, and the largest concentration of these glands is found in the preauricular region and in the postauricular skin along the hairline and down into the mastoid process and neck.[2]

The skin lining the cartilaginous portion of the exteral auditory canal is thicker than that lining the osseous portion, shows very little subcutaneous tissue, and is firmly attached to the perichondrium. There is a fairly well marked layer of stratum corneum and definite papillae may be visualized. No eccrine glands can be observed in histological preparations[8,9] or in sweating experiments.[2,10] The sebaceous glands attached to the hair follicles are exceptionally large, and most of the apocrine glands may be seen draining into the apo-pilo-sebaceous units.[9,11] In the osseous canal the skin is thin and adheres to the periosteum. There is an absence of papillae, little dermis and no subcutaneous tissue. Apocrine and sebaceous glands are rarely found.

Let us consider the histology of these epidermal glands. The sebaceous glands lie superficial in the dermis and consist of multilobulated grape-like glands, all of them opening through short excretory ducts into the lumen of the hair follicle (follicular canal). The excretory ducts are lined by stratified squamous epithelium, which is continuous with the external root sheath of the hair follicle and with the Malpighian layer of the epidermis. Most of the polyhedral cells making up the alveoli of the sebaceous glands are filled with fat droplets. As one progresses towards the duct, the nuclei are seen to shrink and then disappear as the cells break down into the fatty detritus which forms the sebum and which ultimately mixes the apocrine secretion and the horny exfoliated protein-like scales in the follicular canal.

There is no direct evidence for a secretory innervation of the sebaceous glands at the present time. It has been proposed that emotional tensions increase sebaceous gland activity by way of direct nervous stimulation.[12] It has been further suggested on the basis of morphological data that the sebaceous glands are supplied by nerves which form a plexus on the basement membrane of the glands; but there is no proof that these unmyelinated fibers actually enter the parenchyma or that they stimulate glandular activity.

It has been reported that sebaceous gland activity is influenced by various factors such as the chemical composition of the secretion,[13,14] atmospheric temperature, etc. The melting point of sebum is around 30° C; at higher temperatures the sebum is liquid, while at low temperatures it is solid and hard. When fatty acids prevail in the mixture the melting point is low; it is high when the mixture contains cholesterol and wax alcohols. Various investigators[15,16] have stated that the main factor influencing the spread of sebum is moisture on the skin surface. The spread is said to be 140 times greater on moist skin than on dry skin. This would permit the sebum to wash away on hot, humid days leaving the surface vulnerable to maceration and infection.

The apocrine glands are located most abundantly in the skin of the second and third quarters of the external auditory canal and according to von Tröltsch[17] extend along the su-

perior wall almost to the tympanic membrane. Alzheimer[18] reported a great concentration of apocrine glands both on the superior and inferior walls. The apocrine glands develop as outgrowths of the outer sheath of the hair follicles (in contrast to the eccrine sweat glands which develop as direct downward projections from the epidermis). In human ear canals the highest percentage of the apocrine excretory ducts open into the follicular canal while a small percentage of these glands open independently upon the surface of the skin of the ear canal.[9,11]

As shown by Shelley and Hurley in observations on axillary apocrine glands,[3,4] apocrine sweat is a milky whitish or grayish, sometimes yellowish secretion, which forms a light colored cone over the poral surface. It takes the form of an adherent cap which then spreads over the perifollicular skin in a thin film. Autonomic innervation is easily demonstrable. After painful stimuli, the local injection of epinephrine, or intense emotional reactions, a droplet may appear after a latent period of 15 seconds or more. Once a droplet has been secreted, apocrine function ceases for several hours, and further stimulation is ineffective. Apocrine glands do not appear to respond to thermal stimulation.

It seems clear that these glands have an important role in washing the more viscid sebum from the hair follicle and mixing with it to form an adherent protective surface coating. It is worth emphasizing, as presented by Rothman[12] that the stratum corneum offers little or no barrier to infection since it is perforated by the apo-pilo-sebaceous units and thus allows ready contact between external agents and living cells of the sebaceous and apocrine glands. The waxy secretion, when present on the surface and within the follicular canal, interferes with the permeation of noxious agents through the epidermis.

With this knowledge of the histology and the physiology of the skin of the ear canal as a basis, let us proceed to a consideration of the pathogenesis of diffuse external otitis. In our clinical studies,[19] we have had the opportunity of observing large numbers of normal Air Force and Naval volunteers in hot, humid environments. Among these healthy young

adults there was a consistent reduction or absence of cerumen in the lumen of the ear canal. It is our opinion that the oily sebaceous secretions of the apo-pilo-sebaceous units had been carried away as a result of the low viscosity of the sebum at these temperatures and the rapid spread of the lipids on the very moist skin surfaces. This defatting of the skin was facilitated by frequent swimming, diving and under-water activity.

Another factor in the production of dry, scaly ear canals is the accepted practice among parents of removing ear wax from their children's ears. Thus, over-conscientious mothers make a daily ritual of digging at the ear canals. This habit is carried over into adulthood and constant digging at ears and removal of the protective wax covering may become a nervous habit practiced by adults even in the absence of an accumulation of ear wax.

It is conceivable that as a result of continued exposure to intense heat and humidity and poor aeration of the ear canal that the skin of the ear canal may show changes such as a shift of the pH toward the alkaline side,[20] marked hyperkeratosis and plugging of the apo-pilo-sebaceous units. The unprotected upper layers of the stratum corneum then absorb moisture and become swollen and macerated so that proper cornification does not occur.

This is the condition we recognize as the pre-inflammatory stage of external otitis.[21] If, at this time, the patient avoids further exposure to high temperatures and humidity and experiences no further local trauma by water or irritating medications, the hyperkeratosis may disappear in approximately two to three weeks through normal desquamation. On the other hand, if before this hyperkeratosis disappears, there is further exposure to high temperature and humidity, or the patient traumatizes the skin surface, the inflammatory state which we call acute external otitis is produced.

Biopsies from patients with mild external otitis show variable hyperkeratosis, acanthosis, inter and intra-cellular edema. Moderate keratin plug formations are present in the apo-pilo-sebaceous orifices. Changes can be seen in the dermis, which

include subepithelial infiltration of leucocytes, dilatation of blood vessels and perivascular infiltrate especially in the superficial dermis.

As a result of the severe itching or mild pain seen in the early stages of external otitis, irritating medications are introduced into the ear canal. Since the important anti-bacterial and antifungal agents produced by the epidermal glands are not present, rapid growth of fungi and Gram-negative bacilli takes place. If the acute process is not controlled, more severe clinical and pathological changes occur.[22]

On magnification the orifices of the apo-pilo-sebaceous units may be seen producing the cobblestone effect especially visible on the superior and inferior canal walls. The total thickness of the skin is greatly increased by cellular proliferation and edema. There is marked acanthosis with broadening and elongation of the rete pegs. The stratum corneum shows striking changes with multiple fluid-filled spaces and occasional micro-abscesses. Irregularly arranged masses of parakeratotic cells are seen filling the crypts of the hair follicles. The dermal changes include marked vascular changes such as reduction or occlusion of vascular lumina. Contiguous fibrous tissue shows infiltration with eosinophiles and neutrophiles. The infiltrate now becomes superficial and deep, and in some cases dense infiltration of lymphocytes and monocytes are observed around small microabscesses. The apocrine glands may be embedded in exudate, and in some cases leucocytes are found within the lumina of the glands. The sebaceous glands are surrounded by edema and exudate.

As the process progresses, the skin is flooded with infiltrate and superficial and deep microabscesses are produced. These are seen to be true collections of leucocytes within the epidermis or in the dermis, which eventually break through the surface barrier. Such microabscesses appear grossly as single pustules or papules and are often misdiagnosed as furuncles.

Finally, as a result of the inflammatory process and the failure to eliminate the obstruction of the ducts, variable degrees of vacuolization and degeneration of the epithelium of the apocrine glands occur. Evidence of destruction of the

acini of the sweat glands and marked periductal and peri-acinar fibrosis are found.

A reversal of the inflammatory reaction takes place with the institution of oral and topical therapy. The superimposed infection is controlled by topical antibiotics. The extensive edema of the epidermis is eliminated rapidly by proper local therapy. Marked exfoliation of the top layers of the epidermis is followed by a slow return to function of the epidermal glands. The deep infection drains onto the surface of the ear canal.

Unfortunately the destroyed apocrine glands cannot be replaced. If a sufficient number of apocrine glands is destroyed the patient becomes susceptible to reinfection whenever the temperature and humidity rise above a given point, and a state of chronic diffuse external otitis may be produced.

To recapitulate, although all the pathologic, physiologic, and chemical evidence has not yet been obtained, the following concept of pathogenesis of diffuse external otitis will offer a working hypothesis on which therapy may be based: As a result of persistent hot, humid temperatures, changes in the epidermis and dermis of the skin of the ear canal are produced in susceptible individuals which make them vulnerable to infection by Gram-negative bacilli and fungi. If recovery does not take place before an additional exposure to a hot, humid environment occurs, the epidermal glands are unable to pour their anti-bacterial and anti-fungal secretions onto the surface of the skin, and as a consequence, a surface infection develops. If the infection is not brought under control, marked histopathologic changes are seen in the epidermis and dermis, particularly around the apocrine glands and the vascular tree.

Based on this hypothesis, therapy should accomplish the following: *1.* Susceptible individuals should use proper prophylactic agents during hot, humid periods; *2.* Therapy should consist of early topical treatment in mild conditions and a combination of local and systemic treatment in the more severe states; *3.* Therapy then should be soothing and palliative during the mild stage and active and curative in the more severe stages.

A. Mild stage.

1. Remove the patient from the hot, humid environment.
2. Irrigate the ear canal with hypertonic saline in order to clean away any keratin plugs, inspissated wax and debris which have collected in the ear canal.
3. Apply to the lumen of the ear canal mild astringent, drying solutions, for example, aluminum acetate packs or Burow's solution.
4. Use non-irritating vehicles as carriers of therapeutic agents; for example, 5 to 10 per cent boric acid in anhydrous lanolin.
5. Apply topically non-irritating antipruritics combined with antibiotics having low index of sensitivity.

B. More severe stages.

1. Remove patient from hot, humid environment.
2. Prescribe, for oral use, broad-coverage antibiotics.
3. When ear canal lumen is sufficiently patent, remove inspissated infected debris from lumen and tympanic sulcus by use of 3 per cent hypertonic saline irrigations and then apply proper topical antibiotics in non-irritating vehicle.
4. Insert anhydrous lanolin or glycerin pack for 24 hours to reduce edema of the stratum corneum.
5. Continue cleansing treatment until there is evidence of a return of function of the epidermal glands.

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ALLERGY IN ITS RELATION TO THE TONSILS AND ADENOIDS.*

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Some 20 to 25 years ago, during the heyday of otolaryngologic surgery, it seemed that the mere presence of tonsils and adenoids often was sufficient reason for their removal. The slaughter of tonsils and adenoids proceeded at a terrific rate until the pendulum of sound judgment swung back from this radical viewpoint to a more conservative and reasonable attitude, and at the present time most throat surgeons require sound and definite indications before advising the operation.

We all know that too frequently the results following tonsillectomy and adenoidectomy are disappointing. This has led to accusations by our medical friends that the E.N.T. doctor is too prone to operate, and to doubt in the minds of the laity as to the real value of the operation itself. Quite often in advising tonsillectomy and adenoidectomy we are told by the parents that Johnny Jones next door had the same operation a year ago, and still is having the same old trouble. This makes it difficult and at times impossible to convince the parents that in the case of their own child the operation is justifiable and necessary for the patient's welfare.

No doubt, many of you have seen this picture, shown here on the screen, and I am sure you all know what it represents. It appeared in one of the outstanding textbooks on otolaryngology as recently as 1947, and is labeled "The Adenoid Face." I have been unable to find the picture in later texts. This suggests the thought that present-day investigators have learned that in addition to hypertrophied adenoids other pathologic conditions also can produce the same facial expression and features.

* Read at the meeting of the Middle Section, American Laryngological, Rhinological and Otological Society, Inc., Detroit, Mich., January 24, 1955.

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One of these conditions is nasal allergy. Allergic sensitization of the nasal membranes can cause the same facial appearance and the same symptoms as an obstructive adenoid. Too frequently the differential diagnosis is missed, thus inviting further criticism by the patient's family and his pediatrician or family doctor. An example of this is shown by the following case report.

A girl, six years of age, was brought to me a year ago with the complaint of frequent head colds, persistent cough, mucoid and mucopurulent nasal discharge and difficulty in breathing through the nose. Her appetite was poor, and she was undernourished and underweight. After trying tonics, vitamins, nose drops and other remedies the child was taken to a throat specialist. After a very brief examination, according to the parents, he announced that the trouble was all due to an obstructive adenoid and advised adenoidectomy. This was done, along with removal of the tonsils, in the month of May.

Soon after the operation the child's condition began to improve and remained quite satisfactory throughout the summer months; however, the symptoms all returned in October of the same year. When I saw the patient three months later she had lost all that she had gained, including several pounds of weight, and I was told that her condition was no better than it had been before the operation. The parents were unhappy, disillusioned, and bitter in their criticism. Almost their first remark was to the effect that they would permit no further experimenting upon their child in the way of surgery.

It was not difficult to determine why the results of the operation were so disappointing. A strong family background of allergy was revealed by the history of the case, the appearance of the nasal membranes was suggestive, the nasal secretion was loaded with eosinophiles and the child was sensitive to house dust. The specific sensitization was suggested by skin tests and proved by the relief afforded by hyposensitizing treatment with dust extract.

Now it is entirely possible in this case that the tonsils and adenoid eventually should have been removed. Certainly the presence of allergy in no wise renders a person immune to disease in the adenoid or tonsils, or in any other organ. In most cases, however, including the one under discussion, it would seem logical first to attempt to relieve the symptoms by control of the allergy, when present, rather than by immediately hustling the patient off to the hospital for an operation. Following adequate allergic treatment the patient should be in much better condition for the operation if it is still indicated, and the end results should be more satisfactory.

The case just reported is not unique, according to my observation. I have seen several similar cases; also, a considerable number of children with nasal allergy are brought to me for tonsillectomy and adenoidectomy with the diagnosis and recommendation for surgery already made by the family doctor. Some of these patients need the operation. Others are relieved of their symptom by allergic management. All of these cases, however, require some diplomacy and tact in explaining to the parents that an operation may not be necessary, due to the presence of another condition which could be responsible for the trouble.

This news must be broken to the parents in such a way that their confidence in their family doctor shall not be disturbed. I have talked over the situation with those doctors who are kind enough to refer their patients to me, and now instead of suggesting immediate operation they explain the various etiologic factors which might be involved in the case, and instruct the parents to bring the child to me for examination and advice regarding the best way of handling the situation. This more conservative attitude promotes better results and better relations for everyone concerned.

Another case required entirely different management before satisfactory results were obtained.

A boy, seven years of age, was brought to me because of frequent attacks of asthma, present since he was four years old. The attacks were milder and much less frequent during the summer months but became more severe in the fall of the year. Throughout the autumn and winter months he had many head colds, frequently associated with sore throat, which seemed to precipitate the attacks of asthma. He had a personal and family background of allergy and was sensitive to a number of foods and inhalants.

I have seen many children with asthma, associated with nasal allergy, who secured relief from the asthma by control of the allergic condition affecting the nose, and I thought at first that this was another case of that type. The boy's nasal condition improved promptly and quite satisfactorily under hypersensitizing treatment with an extract of house dust. Later, however, during the course of treatment, he developed acute tonsillitis and suffered one of the most severe attacks of asthma that he had yet experienced. Both the tonsillitis and the asthma responded quite favorably to antibiotic therapy.

Cultures taken from the tonsils and pharynx showed pure growths of a staphyloccocus. An intradermal test with this organism gave a strongly positive skin reaction, and also induced another severe attack of asthma. The tonsils and adenoid were removed, and treatment with an autogenous staphylococcic vaccine was begun. Nearly a year has passed, and the patient has had no further attacks of asthma. Both the vaccine and the house dust treatment are still being administered.

The question of bacterial allergy is in considerable controversy. No one seems to know much about it; however, my own brief experience, limited to a few cases similar to the one just reported, has convinced me that bacterial sensitization is a constant threat, and an important factor in allergic disease.

Do tonsils and adenoids "grow back"? Not infrequently, following tonsillectomy and adenoidectomy, in children especially, patches of lymphoid tissue will appear in the tonsil fossae or in the pharynx within a few months after operation. These lymphoid follicles may become sources of infection, or may block the mouth of the Eustachian tube and cause impairment of hearing. The presence of these growths has led to the rather widespread notion that tonsils and adenoids may recur, or "grow back," after removal. Also, many of these patients have been told that the surgeon did a poor job and failed to remove all of the tonsil and adenoid tissue. I am sure that practically every throat surgeon, no matter how skilled, has had this experience in cases in which he was positive that he had performed a thorough operation.

A very plausible explanation of this condition has been advanced by Dr. Norman W. Clein,[1] of Seattle. In a rather extensive survey Dr. Clein found that the great majority of these cases occurred in children who had allergic disease of the nose which had not been diagnosed nor treated prior to tonsillectomy and adenoidectomy. The survey showed that of the untreated allergic children, who underwent this operation, 27 per cent suffered post-operative recurrence of lymphoid tissue, while the incidence of regrowth in those cases having

adequate allergic management prior to operation was only 3 per cent.

Dr. Clein thinks these lymphoid tissue growths are due to allergy, and states that "these findings should always suggest an allergic background." "Allergic disease," he says "predisposes to hyperplasia of lymphoid tissue, especially in the nasopharynx." In my own observation, in the great majority of such cases the development of these post-operative lymphoid patches has occurred in allergic individuals.

In closing, I would like to leave the thought that all candidates for tonsillectomy and adenoidectomy should be more thoroughly screened, and the presence of allergic disease either ruled out or controlled by proper management, prior to operation.

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THE AMERICAN OTORHINOLOGIC SOCIETY FOR PLASTIC SURGERY, Inc.

A short course on Plastic Surgery of the Head and Neck will be given by The American Otorhinologic Society for Plastic Surgery at the Morrison Hotel, Chicago, Illinois, extending from October 7 to October 9, 1955, inclusive. For details write Dr. Louis J. Feit, 73 Park Avenue, New York, N. Y.

PORTUGUESE OTORHINOLARYNGOLOGICAL SOCIETY.

The Sociedade Portuguesa de Otorrinolaringologia e de Bronco-Esofogologia has recently been organized and held its first meeting March 20, 1955, in Lisbon.

The officers are: Dr. Alberto Luis de Mendonca, president, and Dr. Antonio da Costa Quinta, Avenida de Liberdade 65, 1° Lisbon, Secretary.

ORGANIC LESIONS OF THE LARYNX PRODUCED BY MIS-USE OF THE VOICE.*

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Durham, North Carolina.

Laryngologists have long admitted that organic lesions of the larynx may be produced by mis-use of the voice, but few among us really know what actually constitutes mis-use of the voice, or how to correct it. Diagnosis and management of patients afflicted with these lesions would benefit enormously from a sound knowledge of the methods and principles of voice production. Even though none of us expect to qualify as teachers of voice, we should train our ears to perceive vocal strain and to recognize the physical and mental symptoms which attend this difficulty. The laryngologist should be able to handle the problem alone in many instances; in others, he may need help from the voice or speech therapist, or from the psychiatrist. Recognition of the beginnings of vocal difficulty will enable him to know when to enlist such aid and how to direct it.

The primary use of the larynx is not as an organ of speech, but as a protective valve. This protective valve, which prevents the entrance of food and fluid into the lung, has three separate levels which are capable of closure. The vocal cords, which form the lowest plane, must be brought into apposition in order to *produce* sound. The ventricular bands, and the epiglottis and aryteno-epiglottic folds, which form the other two planes, must be kept out of apposition or they will *suppress* sound, and spastic dysphonia or spastic aphonia will result. In addition, the muscles of the pharynx are all *constrictors*, and they, and the other muscles of deglutition, tend to close the passage-way through which laryngeal sound must come out. Undue contraction of these muscles results in muf-

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* Read at the meeting of the Southern Section, American Laryngological, Rhinological and Otological Society, Inc., Charlottesville, Va., January 22, 1955.

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fled, throaty, or "swallowed" sounds. Such muscular tensions, which can be recognized by palpation of the neck, produce fatigue and discomfort.

In 1915, Muckey[1] stated a formula for "correct voice production," which called for "non-interference with *1.* The actions of the vocal cords, and *2.* The opening of the tube that leads out from the pharynx, the buccal cavity and the lips." Though this formula sounds simple, some of nature's strongest impulses tend to defeat it. An infinite variety of spastic interference is possible; muscle tension can vary from slight to marked, and it may occur at one or at several levels, anywhere along "the tube that leads out through the pharynx, the buccal cavity, and the lips." Is it any wonder, therefore, that continued attempts to force *voice* production may bring on vocal trouble, and that traumatic, organic change may result?

The physiology and the anatomy of the larynx have been fully described by Joel Pressman[2] and Chevalier Jackson;[3] their works are well worth further study. In a previous article on the recognition and management of vocal abnormalities, I[4] touched briefly on the contribution made by the voice teacher and the speech therapist toward a clearer understanding of the mechanics of voice production. Both the singing teacher and the speech therapist use terms unfamiliar to most laryngologists. A knowledge of these terms is in itself an introduction to the mechanics of voice production and may well lead to an easier understanding of the cause of vocal trouble.

Phonation, according to the teacher of voice, may start with a "soft" or a "hard" attack. Glottal stroke or "coup de glotte" describes an explosive, sudden, or "hard" attack, usually indicating anger or tension. If repeated, this may result in vocal cord irritation and ultimate damage. A "soft" attack is heard in the gradual onset of phonation. It is associated with pleasurable sensations and relaxed well-being. Phonation is concluded when the vocal cords are released. "Release," too, may be soft and gradual, or it may be grunting, explosive, and productive of irritation.

"Range" is the term applied to the tone area covered by an individual voice from the lowest to the highest possible note. Spoken voice normally falls in the lower one-third of the tonal "range." The "chest register" is employed in producing the lowest tones; here, only the outer portions of the vocal bands vibrate. The lighter "head register" produces the highest tones; when this mechanism of voice production is employed, as in the falsetto voice, only the inner edges of the vocal folds vibrate.

The middle range of tones is produced by a mixture of the two preceding "registers," and utilizes the entire width of the vocal cords. "Resonance" is directly due to the shape, size and character of the infraglottic and the supraglottic spaces. The original thin, weak laryngeal tone is modified by these amplifying, resonating chambers, and the ultimate tone, with its own characteristic blend of primary tones, overtones, and partials, has a "quality" that may be unmistakably identified with the individual.

Three simple factors are often stressed as the basis for correct vocal technique: First, a good speaking or singing voice depends upon the support provided by the diaphragm, aided by the abdominal muscles and those of the back. The proper use of the diaphragm, and the controlled force of the column of air thus obtained, does much to eliminate the use of laryngeal and extra-laryngeal muscles to support tone and to balance pitch. Second, the straightness of the spine, and the position of the head, are important to sound production. When the spine is straight, there is a straight pathway out from the larynx, and if the head is slightly flexed, the anterior cervical muscles are relaxed, preventing constriction. The proper position for phonation is the same as the proper position for mirror laryngoscopy (see Fig. 1). The same open pathway that permits the laryngologist to see permits free egress to sound. Third, space in the oropharynx may be enlarged, and a corresponding increase in roundness of tone or resonance may be accomplished by the elevation of the soft palate. The levators of the soft palate are difficult to train, but the patient may be helped to get the idea by yawning, an act which brings them into play.

It is important in vocal analysis to listen to the singing as well as to the speaking voice. One is occasionally amazed to find a good, clear singing voice in a patient whose speech is hoarse, raucous, or even almost totally suppressed. When present, a good singing voice provides a splendid base on

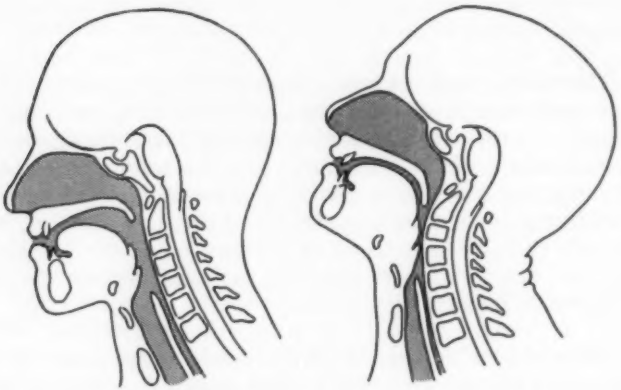

Fig. 1. Showing proper position for phonation with open airway, and improper position producing constriction.

which to begin vocal re-education. When both the speaking and the singing voice are muffled and hoarsened by spastic contraction, it may be difficult, indeed, to effect a cure. The patient may need long study with a competent vocal instructor before improvement can be effected.

The psychiatrist's point of view may also be of help to the laryngologist. All phases of voice production depend upon psychic or emotional balance. The voice is not only characteristic of the individual, but it also varies with his every mood. From it one quickly recognizes the emotional states of happiness, anger, fear, or sorrow. Paul Moses[5] asserts, that in the field of psychiatry many clinical diagnoses can be made simply by listening to the rhythm and quality of the voice.

As laryngologists, we see two major classes of functional voice disorder: the hypofunctional or pseudoparetic type, *i.e.* hysterical aphonia, which *never* leads to organic laryngeal disease, and the spastic or hyperfunctional type, which often does.

Certain types of occupation, as well as certain types of personality, tend to the production of spastic vocal disorders and to ultimate vocal cord damage. Such organic damage is most likely to occur in the person whose occupation and whose personality tend toward the creation of tension. When a high-geared, nervous individual with rapid, explosive, continuous speech is placed in a job which fosters tension, trouble seems inevitable. Patients with some type of spastic vocal disorder are surprisingly frequent in everyday nose and throat practice.

They vary from the factory worker who pitches his voice to speak above noise, to the politician who distorts his voice to speak below his normal range in order to sound impressive. The teacher, the minister, the high pressure salesman, the harried mother of several noisy and overly articulate children, all are candidates for the several varieties of this particular disease. Even the overly articulate children themselves are not exempt.

One needs a practiced ear to recognize the early stage of spastic dysphonia, before organic damage is evident and to sense the true cause of the strained, tired neck and chest muscles, or of the vague discomforts these patients relate to the throat and neck. Even in later stages when diffuse traumatic laryngitis is present, it is often difficult to separate or to distinguish cause from effect. Many such illnesses have been treated vigorously by antibiotic drugs and by voice rest, and most, if not all, have promptly recurred when the rest period ended. All cases of chronic or frequently recurring laryngitis should be suspected of vocal trauma.

Acute vocal traumatic lesions come on suddenly and usually subside promptly. Submucous hemorrhage of the vocal cords is not uncommon in the enthusiastic sports fan, and acute traumatic laryngitis may be induced by similar indiscretions. Lesions of this type usually clear with brief periods

of vocal rest and rarely leave any permanent damage. I have had two singers, however, who have suffered repeated submucous hemorrhage induced by the vocal strain of reaching for a high note. Each of these patients had actual varicosity of vocal cord vessels. Light diathermic coagulation, employing a Walker retinal detachment pin on the end of an insulated rod, served well to prevent further hemorrhage in these patients.

Some of the more chronic organic laryngeal disorders, such as vocal nodule, contact ulcer, and certain types of diffuse laryngitis, appear to be definitely associated with spastic or hyperfunctional disorders, a seeming fact which is difficult to prove. No one as yet has given an adequate explanation for the invariable location of vocal nodule at the junction of the anterior and middle one-third of the vocal cords. The following explanation occurred to me when, at a recent Academy meeting, I saw Dr. Pressman's movie of the sphincter closing mechanism. It showed the vocal cords first coming into contact with each other at the junction of their anterior and middle one-third, then closure of the remaining length of the vocal cords was completed. This was followed by approximation of the ventricular bands, which also began at the junction of the anterior and the middle one-third. Final and absolute closure was completed by approximation of the epiglottis and the arytenoepiglottic folds (see Figs. 2 and 3). It seems possible that partial, spastic closure of the false cords might produce a rubbing or damping action which, repeated many times, could account for local irritation, and ultimate localized thickening of the vocal cords.

Vocal nodule occurs most frequently in workers who must speak above noise. It is a common disorder among the women factory workers in our town; the noise level is high in our tobacco and textile plants, and many jobs require vocalization above it. Its prevention, and sometimes its cure, seems to lie in simple reduction of voice use. This may be brought about, on the part of industry, by insulation and sound-proofing, or by the use of amplifying equipment; until these measures have been accomplished, the patient must learn to speak as little as possible and to move closer to the person to whom he is speaking. Deafness due to industrial noise is now a com-

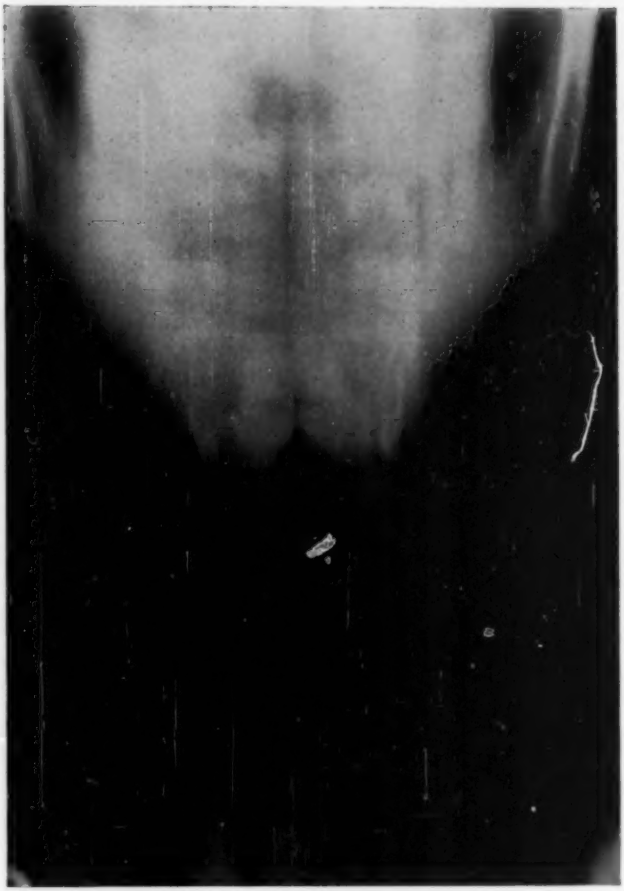

Fig. 2. PA planigram of the normal larynx during phonation, showing vocal cords approximated and ventricular bands relaxed.

pensable disability. Management's increasing awareness of responsibility will undoubtedly result in more active efforts toward noise control.

Vocal nodules vary in appearance at the time of first examination. The more recent ones have a soft, edematous appear-

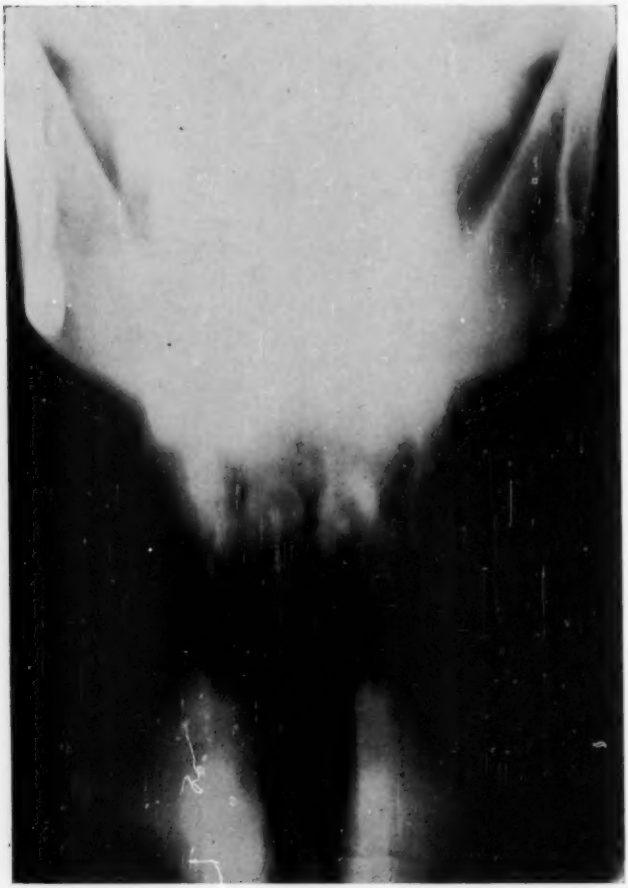

Fig. 3. PA planigram showing spastic dysphonia, vocal cords and ventricular bands both approximated.

ance, while older lesions have the firm, hard appearance of a callus. These older nodules must usually be removed before improvement can be brought about. The soft, edematous type often fade away with simple voice rest. It is obvious that

surgical removal of these nodules is not enough, and that correction of the vocal problems that induce them is essential to prevent recurrence.

Contact ulcer is much less frequent than vocal nodule, and always presents a complicated problem. The patient with this type of lesion usually exhibits the maximum of nervous tension, often coupled with a past history of "nervous breakdown" or of other psychiatric problems. He belongs to a group of "over-doers," mentally, physically, and vocally. The primary lesion here is perichondritis of the vocal process of one or of both arytenoids, and on this there is often superimposed a pyogenic granuloma which may, in some instances, assume a confusingly large size. I remember with chagrin one such patient who presented a cherry-sized posterior lesion which caused hoarseness and mild dyspnea. Our mistake in diagnosis, later corrected, was furthered by the pathologist, who mistook the new-forming capillaries of granuloma for capillary hemangioma.

The treatment of contact ulcer by elevation of vocal pitch, as described by Dr. Peacher,[6] certainly must be accompanied by a very careful psychic and vocal analysis. The "hard" attack of "coup de glotte" would seem to be extremely damaging here. This type of patient, often tense, explosive, and eternally active, requires very careful vocal re-education.

The hard drinking, hard smoking, fast talking salesman is a particular candidate for the type of traumatic, hypertrophic laryngitis which seems most likely to continue to ultimate malignant change. Many of these people, when first seen, show metaplasia of the vocal cords clinically indistinguishable from carcinoma *in situ*. When a period of two weeks of complete abstinence from all three forms of vocal irritation produces no improvement, or when actual progression is noted, biopsy is indicated. I usually employ one of the therapeutic vitamin and mineral preparations and look for general physical abnormalities, especially nephritis and diabetes. It is extremely important that these patients understand the cause of their trouble, since recurrence is frequent unless the new program becomes a permanent part of their way of living.

CONCLUSIONS.

1. Organic lesions of the larynx may be produced by spastic mis-use of the voice. Certain types of occupation, as well as certain types of personality, tend to the production of vocal tension and hence to organic laryngeal damage.

2. Few laryngologists are trained to recognize vocal mis-use. A knowledge of the methods and principles of voice production, together with an understanding of the emotional problems of the patient who tends to mis-use his voice, is necessary to the proper treatment and consequent permanent cure of many of these laryngeal lesions.

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TRAUMATIC TRACHEAL ATRESIA.*

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Injuries in general are occurring with increasing frequency because of the greater emphasis on speed in our land, air, and sea traffic. The following report of an uncommon injury to the trachea may serve to bring to our attention some pertinent points, which can be used in the treatment of the injuries commonly produced.

The patient, a young white girl, 13 years old, was attending a wiener roast on March 14, 1952. One of her companions tossed an unopened bottle of carbonated drink in the fire with a resulting explosion. Flying slivers of glass produced multiple lacerations of the neck in the region of the thyroid cartilage. The patient was taken to the near-by Naval Hospital for emergency treatment. This consisted of hemostasis, treatment of shock, and the insertion of a tracheotomy tube in the wound in the thyroid cartilage.

About 12 hours later, when the patient was in better condition, the wound was explored at the Naval Hospital. The findings were as follows: A laceration extending through the right ala of the thyroid cartilage just below the level of the right vocal cord and extending across to the left arytenoid, an extension of this laceration inferiorly resulting in a 3-4 mm. laceration of the esophagus.

At this time a low tracheotomy was performed, and the wound in the larynx and upper trachea was closed in layers. The wound in the esophagus was not repaired, but a drain was inserted. The patient remained at the Naval Hospital until March 25, 1952, when she was transferred to the E. N. T. service at Roper Hospital by ambulance. At that time she had a small amount of drainage from the laceration in the thyroid cartilage, and air escaped through this on exertion. Saliva was draining from the portion of the wound which extended into the esophagus. The tracheotomy tube was functioning normally. Several days before the transfer a mirror examination of the larynx was reported by the Naval Hospital as revealing the vocal cords moving normally. The patient had only intravenous feedings for five days before the transfer to Roper Hospital.

On admission to Roper Hospital an intranasal feeding tube was inserted and tube feedings were begun. Dr. Edward F. Parker saw her in consultation in regard to her esophageal laceration and suggested lipiodol esophagrams. These were done on March 28, 1952, and repeated on April 1st and April 7th. They revealed a fistulous tract between the esophagus and the trachea opposite the fifth cervical vertebra. This became progressively smaller.

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The patient was discharged from Roper with feeding tube and tracheotomy tube in place on April 8, 1952. She had received penicillin intramuscularly 400,000 units daily, and had an essentially afebrile course. The esophagram was repeated on April 29, 1952, and revealed that the fistula had closed completely. The feeding tube was removed May 1, 1952.

Mirror examination of the larynx while in the hospital revealed a gradually progressing bilateral abductor paralysis of the vocal cords. By April 29, 1952, she had complete fixation of the cords in the midline, and was almost aphonic. It was thought that this progressive paralysis was due to progressive involvement of the recurrent laryngeal nerves with scar tissue from the wound, as the cords were motile when first examined at the Naval Hospital.

She was readmitted to Roper Hospital on May 23, 1952, and a mobilization of the left arytenoid cartilage was done following the Clerf technique and using a horizontal incision. At operation it was found that considerable cartilage and bone had been laid down along the line of the original laceration so that the left arytenoid area was transformed into a solid osteo-cartilaginous strip. Complete mobilization of the left arytenoid was not obtained. The operation was done on May 22, 1952.

The patient developed an abscess near the medial end of the incision after operation. This was incised and drained on May 31, 1952. A fistula between the larynx and neck developed, which closed spontaneously in a few days after insertion of a feeding tube. The patient subsequently made an uneventful recovery. She was discharged home June 7, 1952. Her tracheotomy tube was still in place, but was blocked off part of the time.

At my suggestion the patient saw Dr. Louis Clerf at Jefferson Hospital September 15, 1952. His opinion and suggestions were as follows:

"Your patient apparently has an atresia of the larynx. which I believe is cicatricial rather than paralytic.

"I did a direct examination, but could see no evidence of a lumen.

"I believe that it will be necessary to do a laryngotomy dividing the thyroid cartilage in the midline, removing all cicatricial tissue and securing an obturator. It is important in all of these manipulations to avoid all undue trauma and pressure necrosis. I would suggest that a sponge rubber obturator be employed encased in a finger cot and surrounded by skin graft. The sponge rubber should be secured preferably with stainless steel wire.

"The question of time of its retention in the larynx is difficult to state. I have preferred leaving these in from four to six weeks, when I used a pure gum rubber tube. With sponge rubber the time has been lessened slightly.

"In any event it is going to take a long time to get this young lady well, but it appears to me that the prognosis should be good."

On October 20, 1952, she was re-admitted to Roper Hospital and a thyrotomy was done. On opening the larynx a circular band of scar tissue was found just below the level of the vocal cords, almost completely occluding the tracheal lumen. A direct laryngoscopy done just before the operation had revealed both cords fixed in the midline and no evidence of a lumen. She had been dependent on the tracheotomy tube for breathing for several months before operation, and she had been unable to speak except for a very weak whispered voice.

The band of cicatricial tissue was excised and the edges of the normal mucosa were undermined and approximated using interrupted sutures of No. 0000 chromic catgut.

At the suggestion of Dr. Martin Zwerling of the Naval Hospital, who was my assistant, the right arytenoid cartilage was resected through an incision through the mucosa on the medial aspect of the cartilage. This wound was closed with No. 0000 chromic catgut in continuous interlocking sutures. An obturator of sponge rubber enclosed in a finger cot was laid in place. It extended from just above the vocal cords to about an inch below them. The upper end was fixed in place with a cotton suture passed through the soft tissues of the neck; the lower end, with a cotton suture passed through the tracheotomy wound. The two ends were tied loosely together on the neck. The wound was closed in layers and a dry dressing was applied.

The patient made an uneventful recovery and was discharged home November 4, 1952, with the obturator in place. She was re-admitted November 21, 1952. By using a direct laryngoscope and alligator forceps the obturator was visualized and grasped, the suture was divided and the obturator removed. This was done under intravenous sodium pentothal anesthesia. She made an uneventful recovery and was discharged the next day.

For about the next 12 months she progressed favorably. Her tracheotomy tube was removed after being blocked off for about three weeks. This wound gradually filled in but never closed completely. Her voice improved considerably, most of her phonating being done by the approximation of the false cords. Occasional bouts of acute respiratory infections necessitated hospitalization for inhalations of sodium lauryl sulphate and for penicillin therapy in December 1952.

Again on November 2, 1953, she was re-admitted to Roper Hospital because of nocturnal dyspnea and stridor. This became so marked that it was necessary to re-insert the tracheotomy tube. This was done on the ward under local anesthesia on November 3, 1953. Two days later a bronchoscopy was done, which revealed that the band of scar tissue had reformed just below the level of the cords. A 4x26 Jesberg bronchoscope was the largest one that could be introduced into the narrowed lumen. The patient was discharged on November 7, 1953, with the tracheotomy tube in place. Our plan of therapy was to attempt subsequent dilatations of the larynx, using solid metal laryngeal bougies such as are used to dilate congenital webs of the larynx.

These dilatations were done under intravenous sodium pentothal anesthesia on November 28, 1953, December 12, 1953, January 16, 1954, January 23, 1954 and February 6, 1954. These dilatations apparently accomplished very little, as it was impossible to de-cannulate her, and so treatment was discontinued.

She was not seen again until May 7, 1954, when she came in for a routine check in the office. She had gained about 10 pounds in weight and her voice was somewhat stronger. On mirror examination of the larynx some motility of the right cord was noted. She was still wearing a tracheotomy tube which was blocked most of the time.

At the meeting of the American Academy of Ophthalmology and Otolaryngology in New York in September, 1954, Dr. Joel Pressman's advice was sought. He stated that in his experience, cicatricial rings frequently re-formed after the original excision; however, if a secondary fissure is done and a vinyl plastic tube sutured in the trachea over the site of the scar tissue band the chances of the band forming a third time are rather remote.

Following his suggestion, a secondary laryngofissure was done under general anesthesia at Roper Hospital on September 30, 1954. As was anticipated, a band of scar tissue was present just below the level of the cords. Several longitudinal incisions were made in this band and a portion of a vinyl plastic anesthesia tube was sutured in place with tantalum sutures. The upper suture was brought out through the soft tissues of the neck; the lower one through the tracheotomy wound. Each was tied over a gauze sponge. The wound was closed in layers. The patient made an uneventful recovery, and was discharged four days later on October 4, 1954.

This intratracheal tube was left in place almost seven weeks. It was removed under general anesthesia by means of a direct laryngoscope and alligator forceps on November 10, 1954. The patient was discharged that same afternoon.

The tracheotomy tube was left in place, but completely blocked until December 22, 1954. It was removed on this date, and to date there has been no recurrence of the stridor or dyspnea. Her voice has become much stronger since her final operation. Mirror examination of the larynx shows an adequate glottic chink posteriorly of about 4 to 5 mm., and some motility of the right cord. Phonation is apparently accomplished by the partial approximation of the true cords.

COMMENT AND SUMMARY.

A great amount of new work has been done on surgery of the larynx and trachea, and more is in progress. This case is

presented as a contrast in various methods of surgical approaches to the problem of relieving laryngeal and tracheal atresia secondary to trauma. Clerf's modification of Woodman's operation was first tried but proved inadequate, because a cicatricial band was blocking the airway below the level of the true cords. The cords themselves were anchored in the midline because of cicatricial tissue pinching off the recurrent laryngeal nerves.

The next procedure of laryngo-fissure, excision of the cicatrix, resection of the arytenoid, and the insertion of a foam rubber obturator covered by a finger cot was not the complete answer, because of the secondary formation of the cicatricial band. Dilatation of the band with metal laryngeal bougies failed to give satisfactory results. Finally, a secondary laryngo-fissure, the making of longitudinal incisions in the scar tissue, and the insertion of a vinyl plastic intratracheal anesthesia tube has so far proven satisfactory; however, it is very possible that the larynx may have to be opened again, the scar tissue excised, and a graft applied before a completely satisfactory result is obtained.

96A Bull Street.

BRONCHOESOPHAGOLOGY COURSE

The next Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for the period October 24 to November 5, 1955, under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

THE BLUE EARDRUM: REPORT OF A CASE IN WHICH TREATMENT WAS RADICAL MASTOIDECTOMY.*

KINSEY M. SIMONTON, M.D.,

Rochester, Minn.

Johnson,[1] in 1953, reviewed the literature on the subject of the blue eardrum, otherwise known as idiopathic hemotympanum. He reported one case in detail. In his patient, blood-stained fluid continued to collect in the tympanic cavity after cortical dissection of the mastoid air cells had been done. In conclusion Johnson wrote:

"By a process of elimination, we have arrived at the conclusion that somewhere in the middle ear cleft of these patients exists an area of varicosity, or even hemangioma, which fails to undergo complete tissue repair, but repeatedly breaks down to permit the escape of fresh blood. . . . Some day one of us will exercise the temerity of performing a complete radical mastoid operation upon one of these cases, and perhaps find the true explanation."

In the present paper a case in which idiopathic hemotympanum was treated by radical mastoidectomy is reported. The report is presented in considerable detail, in view of the fact that Johnson found "less than a dozen" references to hemotympanum in the literature.

REPORT OF A CASE.

A white woman 49 years old was first examined in 1944. She complained primarily of pain in the region of the cervical part of the spinal column, and she dated the onset of this pain to a fall 18 months before. Many variable and unrelated complaints suggested nervous instability. Intermittent spasm of the muscles of the right side of the face accompanied exacerbations of her pain. Examination of the ears revealed scarring of the right tympanic membrane. There was no discharge. Perforation was sought, but not found. Tests with tuning forks indicated a combined type of hearing loss on the right. The history indicated that otorrhea had not occurred since the age of eight years.

* From the Section of Otolaryngology and Rhinology, Mayo Clinic and Mayo Foundation. The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

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Examination of the ears in 1946 had produced similar findings.

The patient was examined again in July, 1952. At this time she complained of pain in the right ear. The pain had begun in January, 1952, and had increased progressively in severity. The patient said that anti-allergic therapy had given no relief. Temporary relief of pain had followed aspiration of fluid from the ear. The referring physician said that the fluid was dark brown, and that Papanicolaou stain had revealed no malignant cells. The patient reported that spasm of the muscles of the right side of her face had stopped after the spontaneous discharge of brown fluid from the right ear in April, 1952. Traction applied to the head had resulted in temporary relief of her pain.

Examination of the right ear disclosed marked scarring of the tympanic membrane. The tympanic membrane was dark blue, but did not bulge. Dark brown, sanguineous fluid was removed from the tympanic cavity after myringotomy. Roentgenologic examination of the right mastoid was interpreted as showing partial sclerosis, with decalcification of the remaining mastoid cell walls. Audiometric examination after myringotomy revealed loss of perception in the right ear which averaged 45 decibels through the speech frequencies of 500 to 2,000 cycles per second. Neurologic and general physical examinations disclosed no pertinent changes. Examination of the blood with special reference to coagulation factors revealed no abnormalities.

The right mastoid was explored surgically on July 19, 1952. A postauricular incision was made. The mastoid cortex was seen to be moderately thickened. Some of the mastoid cells contained brown fluid. The mucosa lining the cells was not thickened but was stained brown. The bone forming the intercellular septa was soft and spongy. Free bleeding was encountered. The mastoid cell system was extensive. All visible cells were exenterated. The dura overlying the middle fossa of the skull and the sigmoid sinus were purposely exposed, but this disclosed no abnormality. The superior wall of the bony external auditory canal, including the superior portion of the tympanic ring, was removed. The ossicles were left *in situ*. The limited inspection permitted by this type of exposure revealed no abnormality of the mucosa lining the tympanic cavity. A Lempert type of flap was fashioned, and the ear was closed as in a modified radical mastoidectomy.

Microscopic examination of tissue removed from the mastoid process was reported to show "slight increase in connective tissue around the air cells."

The patient was observed periodically. On September 12, 1952, the mastoid cavity was completely lined with epithelium. At this observation the bluish discoloration of the tympanic membrane was noted for the first time since the operation. Needle aspiration failed to remove fluid from the tympanic space. Repeated observations produced similar findings. The patient complained of intermittent, throbbing pain in her right ear. When she was seen on February 2, 1953, the tympanic membrane was found to be bulging and dark brownish-red. Fluid was obtained by myringotomy and inflation. Relief of pain followed and persisted for three days.

Roentgen therapy was given in April, 1953. A total dose of 800 roentgens was applied in two treatments of 10 mintues each to a field of 10 cm in diameter centered over the right ear. No benefit was noted.

A second operation was performed on July 24, 1953. The mastoid cavity was exposed by an antauricular incision. The skin of the external auditory canal was incised external to the tympanic ring. The tympanic membrane, incus and malleus were removed. Considerable scar tissue was

found around the stapes. The bony wall covering the horizontal portion of the fallopian canal was absent. The facial nerve in this area was enlarged to approximately four times its usual diameter. The mucous membrane lining the tympanic cavity and tympanic extremity of the eustachian tube was removed with exception of that lying in the niches of the round and oval windows. The mucous membrane appeared to be fibrous but otherwise was normal. The tensor tympani muscle was removed from its semicanal. Bleeding during the operation was not excessive. A split-thickness skin graft was applied.

The skin graft failed to take. Epithelization of the tympanic space took place slowly, being complete in ten months. To the time of this writing no discharge has been noted in the right ear since April, 1954. The patient reports that her pain remains.

SUMMARY AND CONCLUSION.

One case of blue eardrum, or idiopathic hemotympanum, is reported. Complete inspection of the mastoid and tympanic cavities was made after radical mastoidectomy. The source of the blood-stained secretions in the ear was not identified. Johnson's hypothesis is not supported by the findings made in examination and treatment of this patient.

REFERENCE.

1. JOHNSON, W. R.: The Problem of the Blue Eardrum, Idiopathic Hemotympanum. THE LARYNGOSCOPE, 63:1096-1117, Nov., 1953.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY HOME STUDY COURSES.

The 1955-1956 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1, and continue for a period of ten months. Detailed information and application forms can be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota. Registrations should be completed before August 15.

UNIVERSITY OF ILLINOIS ANNUAL ASSEMBLY IN OTOLARYNGOLOGY.

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly of Otolaryngology from September 19 through October 1, 1955. This Assembly will consist of two parts:

Part I — September 19 through September 24, 1955, will be devoted to surgical anatomy of the head and neck, fundamental principles of neck surgery and histopathology of the ear, nose and throat. This week will be under the personal direction of Maurice F. Snitman, M.D.

Part II — September 26 through October 1, 1955, will be devoted entirely to lectures and panel discussion of advancements in otolaryngology. The chairman of this section will be Emaneul M. Skolnik, M.D.

Registration is optional for one or both weeks. For further information, address Dr. Francis L. Lederer, 1853 West Polk St., Chicago 13, Ill.

POST-GRADUATE COURSES AT TEMPLE UNIVERSITY.

The following Post-Graduate Courses to be given in this Department during the current year:

Post-Graduate Course in Broncho-Esophagology, June 13-24, 1955; October 17 - 28, 1955.

Post-Graduate Course in Laryngology and Laryngeal Surgery, September 19 - 30, 1955.

These courses are all to be given in the Department of Laryngology and Broncho-esophagology, Temple University Hospital and School of Medicine, under the direction of Dr. Chevalier L. Jackson and Dr. Charles M. Norris. The tuition fee for each course is $250.00. Further information and application blanks can be obtained from Dr. Chevalier L. Jackson, 3401 N. Broad Street, Philadelphia 40, Pennsylvania.

ACADEMY-INTERNATIONAL OF MEDICINE.

A completely revised Fourth Edition of "Professional Films" is now in compilation. (The frequency and number of future insert pages necessary to assure a comprehensive index that is continuously current over a period of years will be determined by the volume of forthcoming productions.) It will include new sections providing biographical data on authors, and information on the audio-visual activities of medical schools, dental schools and post-graduate teaching centers.

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NEW YORK EYE AND EAR INFIRMARY.
ALUMNI ASSOCIATION DINNER.

A subscription dinner honoring Edgar B. Burchell, D.Sc. on the completion of 60 years of distinguished service at the New York Eye and Ear Infirmary, will be given on June 10, 1955, at the 7th Regiment Mess, 643 Park Avenue, at 67th Street, New York City. Cocktails and reception at 6:30, dinner at 7:30 P.M. For further information write to Dr. Joseph H. Krug, Secretary-Treasurer, Alumni Association, New York Eye and Ear Infirmary, 218 Second Avenue, New York City.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Plans have been completed for a joint meeting of the North Carolina Eye, Ear, Nose and Throat Society and the South Carolina Society of Ophthalmology and Otolaryngology at Columbia, S. C., on September 12, 13, and 14, 1955. Headquarters will be the Columbia Hotel.

The following ophthalmologists will be on the program: Dr. E. W. D. Norton of New York, Dr. Frank Carroll of New York, and Dr. William B. Clark of New Orleans.

An announcement of three guest otolaryngologists will be made later.

A most attractive program has been arranged. For further information address Roderick Macdonald, M.D., Secretary and Treasurer, 330 East Main Street, Rock Hill, S. C.

COURSE IN RHINOPLASTY, RECONSTRUCTIVE SURGERY OF NASAL SEPTUM AND OTOPLASTY.

An intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty will be given July 16, 1955, to July 30, 1955, by Dr. Irving B. Goldman and staff at The Mount Sinai Hospital, New York City, in affiliation with Columbia University.

Candidates should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, 5th Avenue and 100th Street, New York 29, New York.

COLBY COLLEGE — AUDIOLOGY FOR INDUSTRY.

Colby College, Waterville, Maine, presents the Third Annual Course in Industrial Deafness, August 7 - 13 inclusive. Objective of the course will be to train personnel in initiating and in conducting conservation hearing programs in noisy industries. Seven full time instructors have been selected from authorities in this field. Class limited to 20 participants.

Registrants will live on the College Campus and the Tuition fee of $200.00 includes board and room. Applications should be made to Mr. William A. Macomber, Director, Division of Adult Education and Extension, Colby College, Waterville, Maine. Frederick Thayer Hill, M.D., Director; Joseph Sataloff, M.D., Assistant Director.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Wm. J. McNally, 1509 Sherbrooke St., West Montreal 25, Canada.
Vice-President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Secretary-Treasurer: Dr. Lawrence R. Boies, 90 S. Ninth St., Minneapolis 2, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Seigniory Club, Montreal, Canada, May 11-12, 1956.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Bernard J. McMahon, 8230 Forsyth Blvd., Clayton 24, Mo.
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Treasurer: Fred W. Nixon, 1027 Rose Building, Cleveland, Ohio.
Librarian, Historian and Editor: Edwin N. Broyles, 1100 North Charles St., Baltimore, Md.
Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

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President-Elect: Dr. Percy Ireland, Toronto, Canada.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

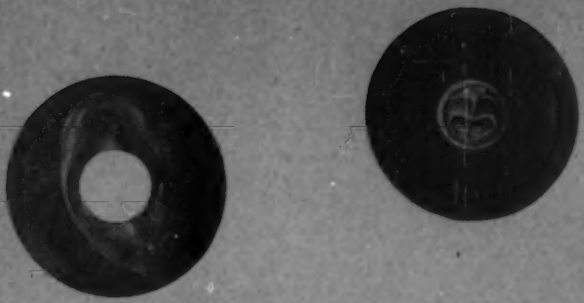
President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October 9-15, 1955.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Daniel S. Cunning, 115 East 65th St., New York 21, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Sheraton Mount Royal Hotel, Montreal, Canada, May 15-16, 1956 (afternoons only).

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October 2-8, 1955.



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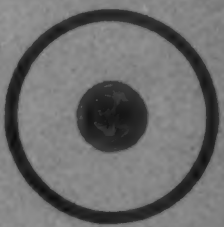

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